The PCB Situation in Germany

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A PCB problem has been known to exist in Germany since 1969. At the Swedish-German Symposium at Baden-Baden, Dr. Jensen, who first published about this problem in 1966, made a report entitled "PCB—the DDT Problem of Industrial Countries."

In September, 1970, there was a PCB conference in Stockholm. One of the reports given there mentioned that a fatal case of biphenyl intoxication had occurred in Finland. The person was a 31-year-old man who had worked in a plant which used biphenyl (not chlorinated) for impregnation. The exposure level was about 100 mg/m^3—10 times the MAC (maximum allowed concentration). He had been working in this industry for 11 years and suddenly developed liver insufficiency—i.e. hepatic necrosis, atrophy, and cirrhosis. General atrophy of the brain cortex was also found. Medical examinations of the other 120 persons working in this plant revealed three additional cases with liver damage (biopsy) and two with abnormal EEGs and impaired peripheral nerve function.

In July, 1970, Acker (University of Münster) was able to find PCBs in human milk at a level of 0.103 ppm, which means 3.5 ppm in the milk fat. In human fat tissue he found an average of 5.7 ppm.

A recent German investigation on rats (Benthe, University of Hamburg) reports the absorption and distribution of PCBs after one inhalation exposure to Pydroul A 200, a mixture of low chlorinated biphenyls. Absorption as well as distribution were studied by measurements of PCB concentration in different organs. By this route very high absorption could be shown. Depending on duration of PCB exposure, a rapid increase in PCB concentration in the liver was observed. After 15 minutes the concentration was more than 50 percent of the maximum concentration attained after two hours (70 μg/g tissue). Concentration in fat after 30 minutes of exposure was 14 μg/g tissue (about 27% of the liver concentration), whereas in brain tissue, only 9 μg/g tissue (17% of the liver concentration) was found at that time.

Depending on the amount of time which passed after the end of aerosol exposure, a rapid decrease of PCBs in the liver during 24 hours was measured and was accompanied by an increase in brain and fat. In the course of two days brain and liver concentrations fell to a minimum value, whereas in fat, a maximum value was attained (260 μg/g tissue) which remained at a constant level in the subsequent period. There was no toxic fatty degeneration of liver under these experimental conditions.

Seven weeks after exposure to low chlorinated PCBs (Tetrachlor), the compounds were found in the fat of rats. After three weeks, 70 percent of the maximum value was still detectable in the fat, and it should be noted that, in a stress situation, PCBs move from fat tissue into the liver.

In addition to the inhalation route, studies were made of other methods of exposure. The results follow:

After intraperitoneal injection of PCBs, a stimulation of the microsomal enzyme systems which are responsible for the metabolism of drugs was found by measuring the O-demethylation, and the increased enzyme activity persisted for a long time. Even after four weeks, the values were still twice the value of the control animals. Because the microsomal enzymes also metabolize adrenal hormones, an effect on reproductive and adrenal hormones can be expected.

After long exposure (3 inhalations for 3 hours),

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a toxic effect on liver function was found: transaminase activity (SGOT, SGPT) had increased on an average by 50 percent. Parallel to this increase of transaminase activity, the liver fat increased from a normal 3.9% to 8.7%. We consider this a toxic effect.

After a subclinical dose of tetrachlorinated hydrocarbon (a dose which does not cause a measurable amount of liver damage) was given, the application of PCBs caused a high degree of liver toxicity; that means that, in the case of a subclinical liver damage, PCB absorption may be dangerous by causing liver dystrophy.

All these results will be published in detail in the next issue of the *Archives of Toxicology*.